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Association of Electroencephalographic Statistical and Complexity Features with Cognitive Function Under Acute Partial Sleep Deprivation: Evidence from a Numerical Stroop Tas

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ABSTRACT

Background: Sleep deprivation adversely impacts cognitive performance. Despite the growing body of studies on electroencephalography (EEG) analysis, the association between complexity and statistical features across EEG electrodes and cognitive function of individuals has not been completely explored. In this study, we investigated the correlation between these features of EEG signals and the cognitive function of individuals assessed in a numerical Stroop test following acute partial sleep deprivation.

Methods: In this study, 19 male and female participants completed both the NST and EEG on the first day to establish their baseline cognitive and neural activity. During the subsequent 24-hour period, participants were instructed to restrict their sleep to a four-hour window between 3:00 a.m. and 7:00 a.m., remaining awake for the rest of the time. On the following day, they had a resting-state EEG recording which followed by a NST.

Results: After acute partial sleep deprivation, higher numerical Stroop error rates were significantly associated with increased EEG complexity measures, including Higuchi's and Katz's fractal dimensions and sample entropy, particularly in frontopolar, frontal, and temporal regions (strongest at Fp1). Kurtosis showed a positive association only at the temporal site (T7), while skewness demonstrated mixed correlations. Standard deviation was positively related to errors at frontal (Fp2) and parietal (P7) electrodes. Overall, increased EEG complexity and variability were linked to poorer cognitive performance.

Conclusion: Increased EEG complexity and variability, particularly in frontal and temporal regions, may serve as potential biomarkers of cognitive impairment during acute partial sleep deprivation.

Keywords: acute sleep deprivation, electroencephalography, numerical Stroop test, fractal dimension, sample entropy, cognitive performance.

1. Introduction

Sleep is a fundamental biological process that plays a critical role in maintaining optimal cognitive performance and neural homeostasis (1). Previous research has consistently demonstrated that adequate sleep is essential for synaptic plasticity, memory consolidation, and emotional regulation (2, 3). In modern society, however, partial sleep deprivation (PSD) has become increasingly prevalent due to lifestyle demands, occupational pressures, and irregular sleep schedules (4). Partial sleep deprivation, defined as a reduction in sleep duration over a short period, has been shown to impair a range of cognitive functions, including attention, executive control, working memory, and decision-making (5). A growing body of evidence indicates that even mild sleep restriction can significantly degrade cognitive efficiency and increase the likelihood of performance errors (6). Empirical evidence suggests that prolonged wakefulness leads to measurable deterioration in behavioral performance and corresponding alterations in brain activity, highlighting the need for objective neurophysiological markers to assess these changes (7). Understanding the neural correlates of cognitive impairment under sleep deprivation is therefore essential for both clinical applications and the prevention of performance-related errors in high-risk environments (8).

Electroencephalography (EEG) has emerged as a powerful and non-invasive tool for

investigating brain dynamics associated with cognitive processes (9). Due to its high temporal resolution, EEG enables the real-time monitoring of neural activity, making it particularly suitable for studying transient cognitive states such as attention and conflict processing (10). Electroencephalography has been widely used in sleep research to characterize different vigilance states and to detect subtle changes in brain function induced by sleep loss (11). Traditional EEG analyses have primarily focused on linear statistical features, including spectral power across canonical frequency bands (delta, theta, alpha, beta, and gamma), which have been widely associated with different aspects of cognitive function (12). For instance, increased theta activity and reduced alpha power have been consistently reported as markers of fatigue and reduced alertness (13). In recent years, there has been growing interest in incorporating nonlinear complexity measures into EEG analysis to better characterize the underlying dynamics of brain function (14). Complexity metrics—such as entropy-based measures, fractal dimensions (FD), and long-range temporal correlations—provide insights into the irregularity, adaptability, and information-processing capacity of neural systems (15). These approaches are particularly valuable because brain activity exhibits inherently nonlinear and nonstationary characteristics that cannot be fully captured by traditional linear methods (16). These measures are particularly relevant in the context of sleep and sleep deprivation, where alterations in neural complexity have been observed (17). For example, studies have demonstrated that EEG complexity reflects the dynamic organization of brain activity and may offer a more comprehensive understanding of sleep-related processes than conventional linear metrics (18). Furthermore, reductions in long-range temporal correlations during sustained wakefulness suggest that sleep deprivation disrupts the critical dynamics necessary for optimal cognitive functioning, thereby impairing processes such as decision-making and working memory (19).

The integration of statistical and complexity-based EEG features has the potential to provide a more holistic characterization of brain function, particularly under conditions of cognitive stress such as sleep deprivation (20). While statistical features capture oscillatory activity and band-specific power distributions, complexity measures reflect higher-order interactions and nonlinear dynamics within neural networks (21). Combining these complementary approaches may enhance the sensitivity and specificity of EEG-based biomarkers for cognitive impairment (22). Recent studies have emphasized that multimodal feature integration can significantly improve the classification and prediction of cognitive states compared to single-feature approaches (23). Despite this potential, relatively few studies have systematically investigated the relationship between these two classes of features and their joint association with cognitive performance, especially in the context of acute PSD.

To evaluate cognitive function under controlled experimental conditions, the Stroop task has been widely employed as a paradigm for assessing executive function, selective attention, and cognitive control (24). The Stroop effect, characterized by increased reaction times and higher error rates, reflects the cognitive effort required to resolve conflict and inhibit automatic responses during the task (25).

Neuroimaging and electrophysiological studies have consistently linked Stroop task performance to activation in the prefrontal cortex and anterior cingulate cortex, regions that are highly sensitive to sleep deprivation (26). Importantly, sleep deprivation has been shown to exacerbate deficits in Stroop task performance, reflecting impaired cognitive control and reduced attentional capacity (27). Previous studies have reported increased error rates and prolonged reaction times in sleep-deprived individuals during Stroop-like tasks (28). Changes in EEG activity during such tasks under sleep-deprived conditions may therefore serve as sensitive indicators of cognitive decline (29). Despite the growing body of literature on EEG analysis, several gaps remain. First, many studies have focused exclusively on either statistical or complexity features, without exploring their combined predictive value. Second, the majority of research has examined total sleep deprivation rather than acute PSD, which is more representative of real-world conditions. Third, there is limited investigation into how these EEG features correlate specifically

with cognitive performance in tasks that require executive control, such as the numerical Stroop test (NST). Addressing these gaps is crucial for advancing the development of reliable EEG-based biomarkers for cognitive function.

The present study aims to investigate the correlation between statistical and complexity features of EEG signals and the cognitive function of individuals experiencing acute PSD, as assessed by a NST.

2. Materials and Methods

2.1. Participants

This study was conducted with a total of 19 volunteers, including 12 men and 7 women, aged between 22 and 36 years old. The privacy and ethical rights of all human participants were fully respected, and informed consent was obtained prior to participation. All individuals were right-handed, in good physical health, and free from any chronic or acute medical conditions. Participants either had normal vision or used corrective lenses and reported no history of substance abuse, including drug or alcohol dependence. None of the participants were under medication other than dietary supplements. Additional demographic details of the participants are provided in Table 1. The Ethics Committee of Tarbiat Modares University reviewed and approved the ethical considerations of this research (Date: June 2022; Reference Code: IR.MODARES.REC.1401.085).

2.2. Experimental procedure

The experimental procedures were carried out over two consecutive days, spanning a total duration of approximately 25 hours. On the first day (prior to sleep deprivation), participants arrived at the laboratory around 9 a.m. Before beginning the experimental tasks, each participant provided written informed consent and completed demographic information form. Each participant initially performed a 10-minute NST according to the procedure described in a later section (see Figure 1A).

At the end of the first day, participants were instructed to avoid consuming any central nervous system stimulants, such as coffee, energy drinks, tea, or other caffeinated products, for the following 24 hours. They were also advised not to nap or engage in strenuous physical activity and to restrict their sleep to a 4-hour period, specifically from 3:00 to 7:00 a.m. Participants returned to the laboratory at 9 a.m. the next morning. No direct monitoring was performed by the experimenter; instead, participants confirmed their compliance with the sleep restriction protocol by completing a brief self-report questionnaire on the second day. After completing the questionnaire, a 3-minute resting-state EEG recording was obtained, followed by administration of the NST (see Figure 1B).

The baseline data collected on the first day prior to sleep deprivation were categorized as the non-sleep-deprived condition (“no SD”). The data obtained on the second day, after PSD, were classified as the sleep-deprived condition (“SD”).

Table 1. Demographic characteristics of the subjects. Mean±SEM.

	Number	Age (years old)	Weight (kg)
Total	19	27.31±1.26	75.00±7.08
Male	12	27.86±1.908	75.57±11.62
Female	7	26.67±1.726	74.33±8.441

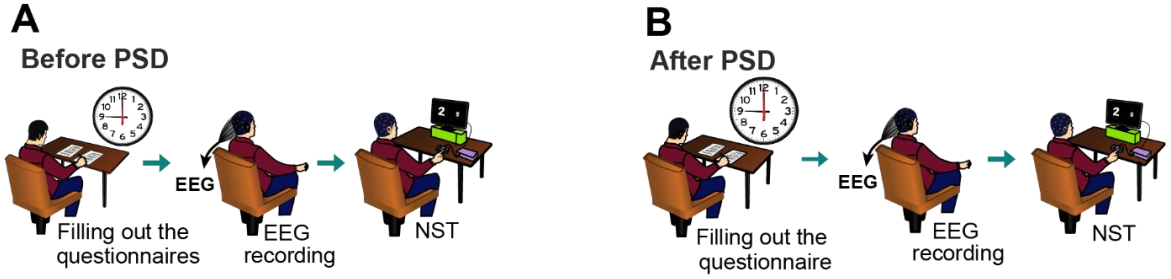

Figure 1. Experimental design. (A) Tasks administered on the first day (before sleep deprivation). (B) Tasks administered on the second day (following sleep deprivation). NST, numerical Stroop test; PSD, partial sleep deprivation.

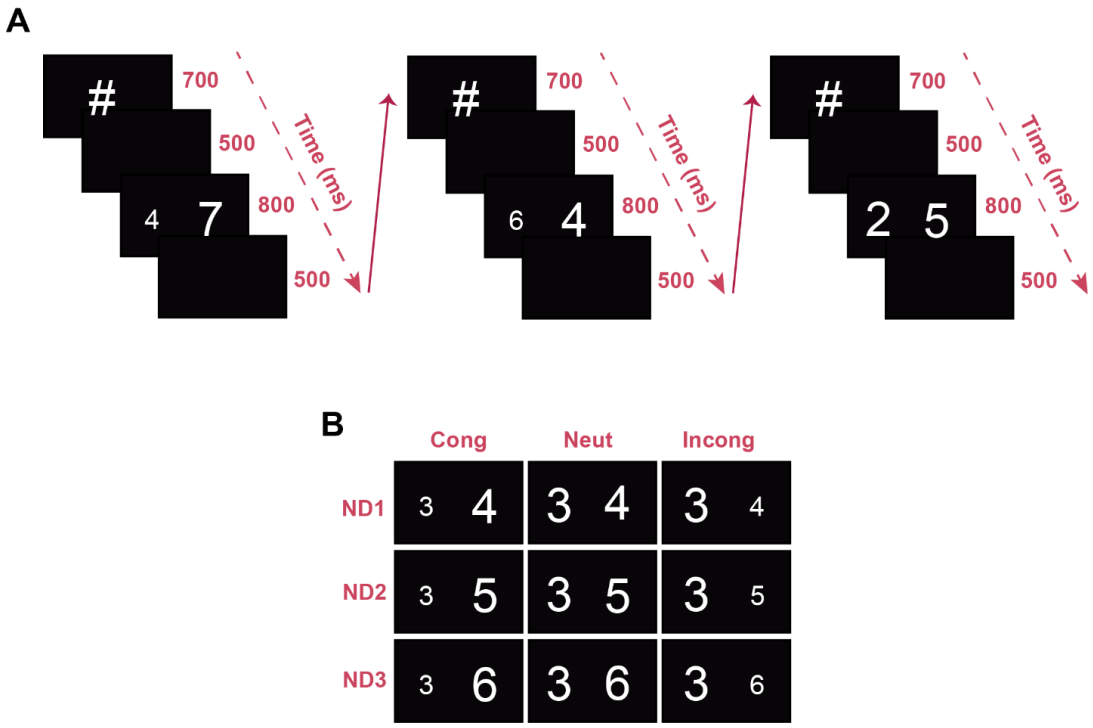

Figure 2. Presentation of stimuli for the NST. (A) The timing protocol for displaying the numbers during the NST trials. (B) Illustration of stimuli displayed across three numerical distance (ND) levels and three congruency conditions: (Cong, congruent; Neut, neutral; Incong, incongruent)

2.3. Numerical Stroop Test (NST)

The NST was employed to evaluate participants' cognitive performance and their ability to inhibit automatic or habitual responses. During each trial, participants concentrated on a 16-inch monitor positioned roughly 80 centimeters away, which presented two single-digit numbers. Each trial began with a hashtag symbol displayed for 700 ms, followed by a 500 ms blank screen. Then, a pair of numbers appeared in white on a black background for 800 ms, after which the screen again remained blank for 500 ms before the next trial began (see Figure 2A). The numbers, ranging from 1 to 9, were shown in Arial font. Participants were instructed to press the key corresponding to the numerically larger digit using their right index finger, disregarding any differences in physical size. The side displaying the larger number alternated randomly. The numerical difference between each pair of digits varied from 1 to 3. The number pairs were presented under three experimental conditions: congruent, where the numerically larger digit also appeared physically larger; neutral, where both digits had the same physical size but differed numerically; and incongruent, where the numerically larger digit was physically smaller (see Figure 2B). A total of 252 trials were conducted, evenly distributed across numerical values, and arranged in a semi-randomized order. Each NST session was divided into two parts, each containing 126 trials, with a five-minute break between sessions to minimize mental fatigue. Finally, the error rate of subjects was calculated as the percent of total trials with incorrect responses.

2.4. EEG recording and pre-processing

During the EEG recording sessions, participants were seated comfortably in a chair with their eyes open. Data were collected using an EEG system (model V.L430, Liv Intelligent Technology, Iran) equipped with active electrodes positioned according to the international 10–20 electrode placement system. Signals obtained from 16 channels, specifically Fp1, Fp2, F3, F4, F7, F8, C3, C4, T7, T8, P3, P4, O1, O2, P7, and P8 were analyzed. The ground and reference electrodes were connected to the right and left mastoids, respectively. Electroencephalography signals were recorded with a low-pass filter set to 100 Hz and sampled at 250 Hz, while electrode impedance was kept below 10 kΩ throughout the sessions.

The acquired EEG data were imported into MATLAB for preprocessing using the EEGLAB toolbox (version 2023.0; sccn.ucsc.edu/eeglab). Initially, baseline correction was applied,

followed by the use of a 48–52 Hz notch filter to remove electrical line noise. Subsequently, a band-pass filter between 1 and 100 Hz was implemented using a zero-phase linear finite impulse response (FIR) filter. After visually inspecting the data and discarding segments containing artifacts, independent component analysis (ICA) was performed with the "runica" algorithm to eliminate unwanted signal sources such as eye blinks, eye movements, muscle activity, and cardiac artifacts. Between two and five components were removed to optimize the overall quality of the EEG recordings.

2.5. Signal processing

The FD was employed to quantify the level of complexity in EEG signals. As a time domain characteristic, the FD has proven effective in differentiating EEG patterns recorded under varying conditions or mental states. Numerous algorithms have been proposed to estimate the FD, among which Higuchi's and Katz's methods are most widely utilized.

Higuchi's algorithm calculates the FD using a stepwise approach. Initially, subsample sets (X_k) are extracted from the main time series (X) according to the following equation:

$$X_k^m = \{X(m+ik)\}_{i=0}^{[\frac{N-m}{k}]}$$

Here, k varies from 1 to k_{max}, m ranges from 1 to k, and N denotes the sample length.

Subsequently, the length of each subsample, denoted as $L_m(k)$, is determined as:

$$L_m(k) = \frac{\sum_{i=1}^{int(\frac{N-m}{k})} |X(m+ik) - X(m+(i-1)k| \times (n-1)}{k \times int\left[\frac{N-m}{k}\right]}$$

Finally, the fractal dimension (D) of the signal is obtained from the relation:

$$\langle L(k) \rangle \propto k^{-D}$$

where ⟨L⟩ represents the average of $L_m(k)$. In this study, EEG segments of 80 seconds were analyzed using k_{max}=8, following established parameters.

Although Higuchi's algorithm is generally more accurate than Katz's, it is notably sensitive to noise interference. To enhance reliability, the Katz method was also applied to estimate FD. This approach involves calculating: (a) the total path length (L), (b) the mean distance (α) between successive points, and (c) the maximum distance (d) between the initial point and all other points in the time series. The FD is then determined as:

$$FD = \frac{\log(\frac{L}{\alpha})}{\log(\frac{d}{\alpha})} = \frac{\log(n)}{\log(n) + \log(\frac{d}{L})}$$

where *n* represents the ratio of *L* to *α*.

In addition to FD, sample entropy was computed to further assess EEG signal complexity. This measure, introduced by Richman and Moorman (2000), refines the concept of approximate entropy by providing more consistent estimates. A higher entropy value reflects greater signal irregularity and a higher degree of complexity. Sample entropy depends on parameters such as the embedding dimension (*m*), the tolerance threshold (*r*), and the total data length (*N*), and is defined as:

$$SaEn\ (m,r,N) = -ln[\frac{A^m(r)}{B^m(r)}]$$

Here, $B^m(r)$ denotes the probability that two sequences match for m consecutive points, while $A^m(r)$ represents the probability that the same sequences also match at the next point (m+1). These probabilities are derived using a relative frequency method. The embedding dimension should be sufficiently large to capture the dynamic structure of the EEG signal, while the tolerance parameter r must balance sensitivity to underlying patterns with resistance to noise. In this study, standard EEG parameters were used: m=2 and r set to 20% of the standard deviation of the 30-second signal window.

It is important to note that FD and sample entropy are distinct yet complementary mathematical tools for evaluating EEG signal complexity. The FD assesses how a waveform occupies space across different scales, capturing its self-similar or fractal characteristics. In contrast, sample entropy measures the degree of irregularity or unpredictability within the signal, reflecting how likely it is that similar data patterns remain consistent as new points are added. When used together, these two measures provide a more comprehensive perspective on the intricate dynamics and complexity of brain activity reflected in EEG signals.

To examine the statistical features of the EEG signal, the kurtosis, skewness, and standard deviation were calculated. Skewness and kurtosis

are measures used to evaluate the degree of dispersion and shape of data distribution. In statistics, skewness indicates the extent of symmetry or asymmetry in a probability distribution, whereas kurtosis reflects the height or sharpness of the distribution curve. In other words, kurtosis quantifies the peakedness of the curve at its maximum point. The standard deviation reflects the extent of variability or dispersion of the EEG signal values around their mean. A higher standard deviation indicates greater fluctuation or instability in the EEG amplitude, suggesting increased neural activity variability, whereas a lower standard deviation represents more stable or consistent brain signal patterns. The kurtosis, skewness, and standard deviation functions were used, respectively, to compute these statistical parameters.

Where applicable, the percentage change in these variables attributable to PSD was calculated as follows:

$$\text{Percentage change} = \left[\frac{\text{Value in desired state} - \text{Baseline value}}{\text{Baseline value}}\right] \times 100$$

2.6. Statistical analysis

All statistical analyses were performed using GraphPad Prism software (version 8, GraphPad Software, Boston, Massachusetts, USA). To assess the relationships between the statistical and complexity features of EEG and NST parameters, linear regression analysis was applied. Additionally, the Pearson correlation coefficient was computed to determine the strength and direction of these associations. Statistical significance was determined with a P-value below 0.05.

3. Results

3.1. Correlation between EEG Higuchi's FD and error rate

The correlation analysis between EEG Higuchi's FD and NST error rate following acute PSD showed electrode-specific effects. A strong, statistically significant positive correlation was observed at Fp1 (r=0.6079, P=0.0058), indicating that higher Higuchi's FD in the left frontopolar region was associated with increased error rate. There were also positive correlations at F7 (r=0.3179, P=0.0448), C4 (r=0.4306, P=0.0457), and T7 (r=0.4870, P=0.0344), suggesting frontal, right central, and left temporal contributions to performance errors after sleep deprivation.

Table 2. Correlation between EEG Higuchi's FD and error rate in the NST following acute PSD for different electrodes. * P < 0.05; ** P < 0.01

Electrodes	r	P-value
Fp1	0.6079	0.0058**
Fp2	0.2978	0.2156
F7	0.3179	0.0448*
F3	0.2621	0.2783
F4	0.3032	0.2071
F8	0.2294	0.3447
C3	0.04475	0.8557
C4	0.4306	0.0457*
P7	0.1533	0.5309
P3	-0.09475	0.6996
P4	0.1324	0.5889
P8	0.2173	0.3716
O1	-0.03848	0.8757
O2	0.04558	0.8530
T7	0.4870	0.0344*
T8	0.07606	0.7569

All other electrodes did not show significant associations with error rate (P>0.05). Overall, the results point to localized frontopolar, frontal, central, and temporal Higuchi's FD performance relationships in the acute PSD condition.

3.2. Correlation between EEG Katz's FD and error rate

The correlation analysis between EEG Katz's FD and NST error rate following acute PSD revealed several region-specific associations. Significant positive correlations were found at Fp1 (r=0.4208, P=0.0428) and Fp2 (r=0.3863, P=0.0423), indicating that higher Katz's FD in bilateral frontopolar areas was related to higher error rates. A further significant positive relationship was observed at F3 (r=0.3171, P=0.0460), suggesting a left frontal contribution to performance errors after PSD. In posterior regions, P8 (r=0.4618, P=0.0465) and T7 (r=0.3427, P=0.0410) showed a significant positive correlation, implying that increased Katz's FD in right parietal and left temporal was also associated with worse NST performance.

All remaining electrodes did not exhibit significant correlations with error rate (P>0.05).

Table 3. Correlation between EEG Katz's FD and error rate in the NST following acute PSD for different electrodes. * P < 0.05

Electrodes	r	P-value
Fp1	0.4208	0.0428*
Fp2	0.3863	0.0423*
F7	0.2839	0.2388
F3	0.3171	0.0460*
F4	0.2423	0.3175
F8	0.1005	0.6823
C3	0.1149	0.6394
C4	0.1536	0.5302
P7	0.2740	0.2563
P3	-0.09719	0.6922
P4	0.2068	0.3955
P8	0.4618	0.0465*
O1	-0.03631	0.8827
O2	0.1364	0.5778
T7	0.3427	0.0410*
T8	0.2347	0.3335

Table 4. Correlation between EEG sample entropy and error rate in the NST following acute PSD for different electrodes. * P < 0.05

Electrodes	r	P-value
Fp1	0.3810	0.0476*
Fp2	0.1649	0.5000
F7	0.4596	0.0477*
F3	-0.001024	0.9967
F4	0.08857	0.7184
F8	-0.2324	0.3384
C3	-0.03713	0.8801
C4	0.2746	0.2553
P7	-0.1142	0.6415
P3	-0.05935	0.8093
P4	0.04080	0.8683
P8	-0.02648	0.9143
O1	-0.06839	0.7809
O2	-0.1656	0.4980
T7	0.3926	0.0463*
T8	0.07490	0.7606

3.3. Correlation between EEG sample entropy and error rate

The correlation analysis between EEG sample entropy and NST error rate following acute PSD revealed a significant positive correlation at Fp1 (r=0.3810, P=0.0476), F7 (r=0.4596, P=0.0477), and T7 (r=0.3926, P=0.0463). These results indicate that higher signal irregularity or complexity (greater sample entropy) in the left frontopolar, left frontal, and left temporal regions was associated with a higher error rate on the NST after sleep deprivation.

All other electrodes showed no significant association with error rate (P>0.05).

3.4. Correlation between EEG kurtosis and error rate

The correlation analysis between EEG kurtosis and the error rate in the NST revealed no significant associations across most electrode sites. However, a significant positive correlation was observed at the T7 electrode (r=0.5182, P=0.0230), suggesting that higher kurtosis values in this left temporal region were associated with increased error rate during the task.

Table 5. Correlation between EEG kurtosis and error rate in the NST following acute PSD for different electrodes. * P < 0.05

Electrodes	r	P-value
Fp1	-0.0219	0.9288
Fp2	-0.1159	0.6366
F7	0.0232	0.9248
F3	0.2197	0.3661
F4	-0.1891	0.4381
F8	-0.0534	0.8280
C3	-0.0653	0.7905
C4	-0.2945	0.2210
P7	-0.0275	0.9109
P3	0.1270	0.6043
P4	-0.1358	0.5794
P8	-0.0137	0.9557
O1	-0.0629	0.7978
O2	-0.0161	0.9480
T7	0.5182	0.0230 *
T8	-0.0037	0.9880

3.5. Correlation between EEG skewness and error rate

Evaluating the Pearson correlation analysis between EEG signal skewness and the error rate in participants experiencing acute PSD showed significant correlations at four electrode sites comprising Fp1 (r=-0.3180, P=0.0446), Fp2 (r=0.3344, P=0.0417), P7 (r=0.4602, P=0.0474), and T7 (r=-0.4298, P=0.0463). Specifically, Fp2 and P7 showed a significant positive correlation, while Fp1 and T7 demonstrated a significant negative correlation, whereas other scalp areas showed no significant associations (P>0.05).

Table 6. Correlation between EEG skewness and error rate in the NST following acute PSD for different electrodes. * P < 0.05

Electrodes	r	P-value
Fp1	-0.3180	0.0446*
Fp2	0.3344	0.0417*
F7	-0.0731	0.7662
F3	-0.2774	0.2502
F4	0.1222	0.6183
F8	-0.0089	0.9709
C3	-0.0426	0.8624
C4	-0.2178	0.3704
P7	0.4602	0.0474 *
P3	0.2142	0.3786
P4	0.2034	0.4037
P8	-0.0162	0.9475
O1	-0.2048	0.4004
O2	-0.2014	0.4083
T7	-0.4298	0.0463 *
T8	0.0851	0.7291

Table 7. Correlation between EEG standard deviation and error rate in the NST following acute PSD for different electrodes. * P < 0.05

Electrodes	r	P-value
Fp1	-0.2655	0.2720
Fp2	0.3809	0.0477*
F7	0.1293	0.5977
F3	0.2338	0.3353
F4	0.2416	0.3190
F8	0.1857	0.4466
C3	0.0060	0.9805
C4	0.1673	0.4936
P7	0.3662	0.0231*
P3	0.2920	0.2251
P4	0.1393	0.5696
P8	0.2309	0.3415
O1	-0.1165	0.6348
O2	0.1282	0.6009
T7	0.0134	0.9564
T8	0.1985	0.4152

3.6. Correlation between EEG standard deviation and error rate

Examining the relationship between the standard deviation of EEG signals and the error rate during the NST revealed a limited number of statistically significant associations. Specifically, a significant positive correlation was observed at P7 (r=0.3662, P=0.0231) and Fp2 (r=0.3809, P=0.0477) electrodes, indicating that greater EEG variability at these parietal and frontal sites is associated with a higher error rate.

In contrast, the remaining electrodes did not show statistically significant correlations (P>0.05), with generally low or negligible correlation coefficients.

4. Discussion

The present study investigated the relationship between statistical and complexity features of EEG signals and cognitive performance, as measured by error rate in the NST, under acute PSD. The findings revealed a consistent pattern in which increased EEG complexity and variability—particularly in frontal and temporal regions—are associated with poorer cognitive performance. These results provide important insights into the neurophysiological mechanisms underlying executive dysfunction during sleep restriction. These findings are in line with prior studies reporting that sleep deprivation leads to increased neural irregularity and reduced efficiency of information processing, particularly in executive control networks (30, 31).

A finding of this study is the significant positive correlation between Higuchi's FD and NST error rate, especially in the left frontopolar (Fp1), left frontal (F7), right central (C4), and left temporal (T7) regions. Similarly, Katz's FD showed significant positive correlations with error rate in bilateral frontopolar (Fp1, Fp2), left frontal (F3), right parietal (P8), and left temporal (T7) regions. Higher FD values show increased irregularity in brain dynamics. The observed association suggests that excessive neural complexity following PSD may reflect dysregulated or inefficient information processing, leading to impaired cognitive control. This interpretation aligns with previous studies showing that optimal cognitive performance is associated with a balance between regularity and complexity in neural systems, while deviations—either toward excessive regularity or randomness—can impair function (16, 32). Moreover, prior research has demonstrated that sleep deprivation disrupts long-range temporal correlations and critical brain dynamics, supporting the notion that altered fractal properties may underlie cognitive deficits (32, 33). Consistent with the findings of Ivanov et al., (1999) the observed increase in FD may reflect a

breakdown of scale-invariant neural organization under sleep deprivation (34).

The involvement of frontopolar and frontal areas is particularly noteworthy, as these regions are central to executive functions, including conflict monitoring and response inhibition which are key processes in the Stroop task. The findings are consistent with neuroimaging and EEG studies indicating that sleep deprivation disproportionately affects prefrontal cortex functioning, leading to reduced cognitive control and increased error rates (31, 35). The additional involvement of parietal and temporal regions may reflect disruptions in attentional networks and sensory integration processes, which are also critical for performance in NST. This pattern is in agreement with previous EEG and fMRI studies showing that the prefrontal cortex is one of the most vulnerable regions to sleep loss, with compensatory recruitment of parietal areas during cognitively demanding tasks (36, 37).

The results for sample entropy further support the role of increased neural irregularity in impaired cognition. Significant positive correlations in left frontopolar (Fp1), frontal (F7), and temporal (T7) regions indicate that higher signal unpredictability is associated with worse performance. Sample entropy is widely used as a measure of signal complexity and has been shown to increase under conditions of cognitive fatigue and reduced vigilance (38). Previous studies also reported elevated standard deviation of EEG power (particularly in theta band) following sleep deprivation, which can be interpreted as a breakdown of coordinated neural activity and reduced efficiency of information processing (39). Notably, the predominance of left-hemispheric effects may suggest lateralized vulnerability of cognitive control networks, although this requires further investigation. These findings are also consistent with the work of Pincus (1991) and subsequent studies, which indicate that higher entropy values reflect increased randomness and reduced predictability in physiological signals under stress or fatigue conditions (40).

Among the statistical features, kurtosis demonstrated a significant positive correlation with error rate exclusively at the T7 electrode. This suggests that changes in the distribution of EEG amplitudes—specifically the presence of more extreme values—may be linked to impaired temporal lobe function during PSD. The temporal cortex plays a role in integrating sensory information and supporting aspects of attention, and its dysfunction may contribute to increased cognitive errors. Although limited in spatial extent, this finding is consistent with reports that sleep deprivation can induce localized alterations in cortical excitability and signal distribution (41). This localized effect is comparable to previous findings suggesting region-specific alterations in EEG signal distributions rather than global changes under sleep deprivation (42).

Skewness analysis revealed both positive and negative correlations depending on electrode location, indicating region-specific asymmetries in EEG signal distribution. Positive correlations at Fp2 and P7, alongside negative correlations at Fp1 and T7, suggest that PSD induces complex, non-uniform alterations in neural activity. Such asymmetries may reflect imbalances in hemispheric processing. Previous research has highlighted that sleep deprivation can differentially affect hemispheric activity and lead to compensatory mechanisms in certain brain regions (43). This hemispheric asymmetry is in line with earlier studies reporting lateralized brain responses and adaptive compensation following sleep restriction (44).

The analysis of standard deviation, as a measure of signal variability, showed significant positive correlations at Fp2 and P7. Increased variability in EEG signals has been associated with reduced neural stability and increased cognitive noise, which can impair task performance. This is consistent with studies suggesting that sleep deprivation leads to greater moment-to-moment fluctuations in neural activity, contributing to lapses in attention and increased error rates (45). However, the relatively limited number of significant findings for standard deviation suggests that variability alone may not be a robust global predictor of cognitive impairment, but rather exerts localized effects. This observation further supports previous reports that variability-based measures are more sensitive to regional dynamics than global cognitive decline (46).

Taken together, the findings of this study highlight the complementary value of combining statistical and complexity-based EEG features to better understand cognitive dysfunction under sleep deprivation. While complexity measures

(HFD, KFD, entropy) consistently showed strong associations with performance deficits, statistical features provided additional, region-specific insights into neural variability and distributional changes. Importantly, the convergence of results across multiple feature types in frontal and temporal regions underscores the critical role of these areas in sustaining cognitive control during sleep loss. Unlike many previous studies that have focused on a single class of features, the present study integrates both nonlinear and statistical measures, providing a more comprehensive and multidimensional characterization of EEG alterations under sleep deprivation (47).

Despite these contributions, several limitations should be acknowledged. The sample size may limit the generalizability of the findings, and the correlational design precludes causal inferences. Additionally, the study focused on error rate as the sole behavioral measure; incorporating reaction time and other performance metrics as supplementary parameters could provide a more comprehensive assessment of cognitive function. Furthermore, the monitoring of participants' sleep control was not rigorously implemented, which may affect the interpretation of the observed EEG patterns. One additional limitation is the relatively short duration of the analyzed EEG segments (three minutes). Although nonlinear complexity measures such as FD and Sample Entropy have been widely applied to short-term recordings (e.g., 2–5 minutes) in previous studies (40, 48–50, 34), shorter signal lengths may still influence the stability and absolute reliability of these estimates. However, as all analyses were conducted on segments of equal length across participants and conditions, the comparative validity of the results is preserved. Despite this limitation, the observed consistency of results across subjects suggests that the selected segment length was sufficient for capturing the relative differences of interest.

Future research should also explore multimodal approaches and longitudinal designs to better characterize the temporal dynamics of EEG changes during sleep deprivation, potentially including enhanced sleep monitoring protocols.

5. Conclusion

In conclusion, this study demonstrates that increased EEG complexity and variability, particularly in frontopolar, frontal, and temporal regions, are associated with impaired cognitive performance during acute PSD. These findings suggest that integrated EEG features may represent candidate EEG-based markers of cognitive performance alterations associated with acute sleep deprivation; however, further studies with larger cohorts are needed to establish their reliability and potential clinical utility.

Ethical considerations

This study was conducted in accordance with ethical standards and was approved by the Ethics Committee of Tarbiat Modares University (Approval Code: IR.MODARES.REC.1401.085).

Informed Consent Statement

Informed consent was obtained from all subjects involved in the study.

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Conflict of Interest

The authors declare no conflicts of interest.

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Author Contributions

Hanieh Riazi: Acquisition of data, Analysis and interpretation of data, Drafting the article; Amir Shojaei: The conception and design of the study, Revising the article, Final approval of the version to be submitted.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request. The data are not publicly available due to privacy restrictions.

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